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THE DISTRIBUTION OF *BRUCELLA melitensis* VARIETY *melitensis* IN THE UNITED STATES

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REVIEW OF THE LITERATURE

For many years after the recognition of Malta fever in Mediterranean countries, the United States was supposed to be free from that disease, now called brucellosis. The first series of cases in this country was found in Texas.

In 1911 Ferenbaugh reported that he had found 5 cases of Malta fever in Texas. All the patients had been in contact with goats. In the same year Gentry and Ferenbaugh published the results of a more extended investigation in which they diagnosed 7 human cases in addition to the 5 reported by Ferenbaugh. They found that 19.4 percent of the 128 goats which they tested were positive for *Brucella* agglutinins. Their investigations led them to believe that Malta fever had probably been endemic in Texas for at least 25 years.

The next year Yount and Looney reported 5 cases of Malta fever occurring in persons connected with the goat industry in Arizona; and in 1913 Yount reported another case, which was fatal. In that same year Wellman and Eustis diagnosed a case of Malta fever in Louisiana in a patient who had contracted the disease in Texas. They detected Malta fever in this patient by testing for *Brucella* agglutinins a group of serums which had been found to be negative to the Widal test. Among 46 serums tested, they found the one positive for *Brucella* agglutinins. It would seem that this one positive result might have suggested that brucellosis was not a rare disease and that further investigations might yield interesting data; nevertheless, no further significant investigations to find cases of brucellosis were carried out until a number of years later, when a definite outbreak occurred. In 1922 Lake, of the Public Health Service, diagnosed 35 cases of Malta fever in Phoenix, Ariz. He traced the source of infection to the milk of infected goats. From information obtained from local health authori-

ties and physicians he was convinced that the disease had been present in Arizona at least 14 years.

Thus, during the early part of the present century the recognized cases of brucellosis in this country were associated with goats in the Southwestern States (excepting one isolated case recognized by Craig in Washington, D. C., in 1905). It was believed that the human disease was limited to caprine sources in the Southwestern States.

Recently Meyer and Eddie confirmed the incidence of *Brucella* infections in goats of the Southwest. They studied strains isolated from goats received from Phoenix, Ariz., and Carlsbad, N. Mex., and found that they belonged to the *melitensis* variety.

As the years passed, reports were made of the occasional isolation of the *melitensis* variety in various other parts of the country. Tyndale and Viko reported that the State veterinarian of Utah died in 1923 as a result of handling placental tissue from an infected goat. He had gone to the southern part of the State to investigate the goats following the death of two herders believed to have been infected with *Brucella*. *Brucella* were isolated from the urine of the veterinarian, but the strain was lost without having been classified. The caprine origin of the infection, however, and the severity of the disease, suggest that the strain concerned in these three cases must have been of the *melitensis* variety. Meyer (1936) and his collaborators have worked quite extensively in southern Utah, and they found the goats of that region to be infected with the *melitensis* variety.

In 1925 the writer published the results of a serological classification of *Brucella* strains isolated from man and domestic animals. Many of the cultures were from distant areas in the United States. The collection included cultures of the *melitensis* variety isolated (by the investigators already mentioned) from human cases in Texas and Arizona, one culture from an aborted bovine fetus in Maryland, and another from an aborted equine fetus in Iowa. Later, Huddleson studied these strains in respect to bacteriostatic reactions and confirmed the classification in the *melitensis* variety of all excepting the equine strain, which proved to be atypical in that it agreed with the *abortus* variety in its sensitivity to dyes.

Later, in 1925, after the results of the classification study had been published, the writer received serum from a human case of brucellosis in Rocky Mount, N. C. From this serum the *melitensis* agglutinins were only partially removed by absorption with *abortus* antigen. The reaction indicated that the infection was with the *melitensis* variety. A few years later, Huddleson reported that two strains isolated from bovine sources in Michigan belonged to the *melitensis* variety; and a few years after that Carpenter and Boak reported that they had isolated the *melitensis* variety from cow's milk in three widely separated towns in New York State. These scattered observations suggested

that infections with the *melitensis* variety were more widely distributed in the United States than was generally supposed.

In European countries also, cattle have been found to be infected with the *melitensis* variety. Shaw, a member of the British Royal Commission on Mediterranean Fever, reported that he cultivated "*Micrococcus melitensis*" from the milk of cows in Malta. Descriptions of these strains by which they might have been identified with the *melitensis* or *abortus* variety were necessarily lacking. More recently, Taylor, Lisbonne, and Vidal cultivated *Brucella* organisms from cows' milk in France and identified them with the *melitensis* variety according to modern methods.

Taylor and his collaborators, and also Gilles, Pérès, and Gulty, have reported that, in the east of France, where human infections are derived from cattle, the *melitensis* variety is responsible for about 95 percent of human infections, although in the cattle of that region the *abortus* variety is the more common cause of infection.

The infection of cattle with the *melitensis* variety is a matter of concern not only on account of the greater number of human cases which an infected cow may cause, but also on account of the greater severity of the human disease caused by the *melitensis* variety. Molinelli made a comparative clinical study of brucellosis indigenous to two sections of Argentina. In the Andes region the human infections are caused by the *melitensis* variety, and the illnesses were found to be much more severe, the mortality higher, the disease lasting longer, and nervous symptoms more common than in the littoral, where the infections are due to the *abortus* variety of the organism.

THE AGGLUTININ ABSORPTION TEST FOR THE DETERMINATION OF THE PREVALENCE OF INFECTIONS WITH *BRUCELLA MELITENSIS* VARIETY *MELITENSIS*

Incidental to the surveys regarding human cases of chronic brucellosis conducted by the United States Public Health Service in several sections of the United States, an opportunity presented itself to investigate the prevalence of *melitensis* infections in man in the surveyed areas by means of the agglutinin absorption test. The use of this test necessitates a discussion of its reliability in identifying the type of infection.

The most satisfactory method for determining the type of infection in any given case of brucellosis is to isolate the organism and study it. Cultures are not readily obtainable from every case, however, and that is particularly true in chronic cases. The agglutinin absorption technique offers another method of obtaining information as to the type of *Brucella* concerned in any given case.

If two samples of serum from a brucellosis patient are saturated, the one with the homologous antigen and the other with an antigen of a heterologous variety, and then the remaining agglutinins in each sample are tested against both the homologous and the heterologous antigens, a reaction will occur with the homologous antigen in the sample which was absorbed with the heterologous antigen, but no reaction will occur in the sample absorbed with the homologous antigen. The agglutinin absorption test may be carried out on any serum which has a titer of 1 to 160 or higher.

The advantage of the agglutinin absorption test is that it is applicable to cases from which cultures can not be obtained. On the other hand, there are limitations to the information which may be gained by the use of the agglutinin absorption test. It does not distinguish the *abortus* from the *suis* variety, for they behave alike serologically; further, there is a small percentage of *Brucella* strains which are atypical, with irregular correlations between the serological reactions and other characteristics. Since the usefulness of the agglutinin absorption test in distinguishing the *melitensis* from the *abortus-suis* group depends on the rarity of atypical strains, a knowledge of their prevalence is necessary for correct interpretation of data obtained by absorbing agglutinins from patients' serum.

In 1925, when the writer published the results of the serological classification of 68 *Brucella* strains, there was no other known test by which they could be differentiated. Four years later, when Huddleson devised the bacteriostatic tests which have proved so useful for classifying *Brucella*, 44 strains which had been classified by the writer were sent, at his request, for his study of the correlation of the two systems of classification. He reported irregularities in two strains (4.5 percent). One of the irregular strains was a foreign strain of bovine origin; the other was the equine strain from Iowa which has been mentioned previously.

Recently Veazie and Meyer reported the results of their study of 447 *Brucella* strains isolated in various parts of the United States and in foreign countries. Among them were 20 strains too rough to be classified. Among the remaining 427 strains only 26, or 5.8 percent, were atypical, in that they failed to conform both serologically and culturally to either the *melitensis* or *abortus-suis* type. Only 11 of their irregular strains, all of bovine origin, were isolated in this country. Eight of these 11 strains had been isolated from different cows in a single dairy.

Investigators of other countries have reported results similar to the American findings. Olin and Lindström studied 103 strains, the majority of which were from human cases in Sweden. Two strains (1.9 percent) were atypical.

Wilson studied over 300 *Brucella* strains. He divided them into two groups—the main group from many parts of the world, and a smaller group from the northeast, east, and southeast of France. In the main group of 165 strains, only 1 was atypical. In the group of 156 French strains, 41 were too rough to be classified, and a large percentage of the remaining strains gave atypical reactions. Ten subgroups were found. Thus the irregular strains of Wilson's collection were received from one geographical area, where the *Brucella* organism seems to be undergoing a transition.

Disagreeing with the results of other investigators is the report of Francis, who studied 23 strains and reported 10 of them (43.5 percent) irregular. His results cannot be explained on the ground that they came from some restricted locality where, as Wilson and also Veazie and Meyer have shown, atypical strains may be widely distributed. Francis' atypical strains were from 5 different localities in the United States, and one group of 3 bovine strains was from Germany. It is not clear why Francis, who studied a relatively few strains, should have obtained results so different from those of other investigators.

If American strains alone are considered, altogether 259 *Brucella* strains have been studied for correlation between serological and bacteriostatic reactions, with results as shown in table 1. Thus, among the American strains studied, 92.7 percent were typical strains, agreeing in classification when grouped according to bacteriostatic reactions or agglutinin absorption reactions. From this it may be concluded that neither test alone will classify individual strains as belonging to the *abortus-suis* or *melitensis* group with absolute certainty, but that collected agglutinin absorption data will give general information as to the types of infection prevalent in a given locality.

TABLE 1.—Record of atypical *Brucella* strains isolated in the United States

Observer	Number of strains studied	Atypical strains	
		Number	Percent
Evans and Huddleson.....	29	1	3.4
Francis.....	19	7	36.8
Veazie and Meyer.....	211	11	5.2
Total.....	259	19	7.3

AGGLUTININ ABSORPTION TECHNIQUE

In the present study the following technique was used to obtain information concerning the variety of the infecting strain in serums of a titer of 1 to 160 or higher:

The cultures used as absorbing antigens were no. 456 of the *abortus* variety and no. 428 of the *melitensis* variety. They were grown on

1 percent glucose agar in Blake bottles. Each bottle was inoculated with the entire growth from 1 agar slant suspended in about 1.5 cc of saline solution. After 48 hours' incubation the growth was washed off with about 15 cc physiological saline solution containing 0.5 percent formalin, by rocking the bottle in the hands. After standing in the refrigerator a few days the dense bacterial suspension was centrifugalized, the clear supernatant fluid was discarded, and saline solution containing 0.5 percent formalin was added to restore the original volume. This suspension was then standardized to a density equivalent to 20,000 parts per million of the silica standard.

In the earlier study (1925) it was found that an antigen of a density of 60,000 p. p. m. would absorb all homologous agglutinins from a serum with a titer of 1 to 640 when the absorption was carried out in a 1 to 5 dilution of the serum; an antigen of half that density would absorb all homologous agglutinins from a serum with a titer half as high; and an antigen of twice that density was required to absorb all homologous agglutinins from a serum with a titer twice as high. In this study an antigen of a density of 60,000 p. p. m. was always used to absorb serums of a titer of 1 to 640; and the density of the antigen was reduced proportionately to absorb serums of lower titer. Serums of higher titer were diluted to a titer of 1 to 640, and the diluted serum was absorbed with an antigen of a density of 60,000 p. p. m. The procedure was as follows:

An equal quantity of glycerine is added to the serum in the field before sending it to the central laboratory. It was found that the test required 1.4 cc of the serum-glycerine mixture to give sufficient absorbed serum of a 1 to 5 dilution for the test. It requires 10.5 cc of the stock antigen to obtain an antigen of a density of 60,000 p. p. m. to absorb the serum in a 1 to 5 dilution. The tube containing 10.5 cc of the stock antigen was centrifugalized, 8.4 cc of clear supernatant fluid was removed, and 1.4 cc of the serum-glycerine mixture was added to the remaining 2.1 cc of antigen. The sediment was emulsified and the tube was then placed in a water bath at 37° C. After 4 hours it was removed to the refrigerator. The next day the tube was again centrifugalized and the agglutinin titer of the clear supernatant fluid was determined by testing with both the *abortus* and *melitensis* antigens. For this test 0.5 cc of antigen of a density equivalent to 500 p. p. m. of the silica standard was added to each of the series of tubes containing the diluted serum. Protocols, with the data for two serums, are given in table 2.

TABLE 2.—Type of *Brucella* infection as determined by agglutinin absorption tests

Serum	Treatment of serum	Abortus agglutinins serum diluted 1 to—									Melitensis agglutinins serum diluted 1 to—								
		10	20	40	80	160	320	640	1,280	2,560	10	20	40	80	160	320	640	1,280	2,560
Charlotte, 209.	Not absorbed.....	2	3	3	3	3	0	0	-----	-----	2	3	4	3	3	2	0	-----	-----
	Absorbed with <i>abortus</i> . ¹	0	0	-----	-----	-----	-----	-----	-----	-----	4	3	3	0	0	-----	-----	-----	-----
	Absorbed with <i>melitensis</i> .	0	0	0	0	0	-----	-----	-----	-----	0	0	-----	-----	-----	-----	-----	-----	-----
San Anto- nio, 309.	Not absorbed.....	4	4	4	4	4	4	4	4	3	4	4	4	4	4	4	4	4	0
	Absorbed with <i>abortus</i> .	-----	-----	0	0	0	0	0	0	-----	-----	-----	0	0	0	0	0	0	-----
	Absorbed with <i>melitensis</i> . ²	-----	-----	4	3	2	0	0	0	-----	-----	-----	0	0	0	0	0	0	-----

¹ Results indicate *melitensis* infection.² Results indicate *abortus* infection.

RESULTS OF THE PRESENT STUDY

The brucellosis surveys were conducted especially for the purpose of finding chronic cases, which are likely to have serums with a titer of agglutinins too low for the absorption test. Occasionally, however, serums were received at the central laboratory with titers high enough for the test. Table 3 gives the results obtained with the serums from the three survey areas.

TABLE 3.—Prevalence of *Brucella melitensis* variety *melitensis* in 3 survey areas, as indicated by agglutinin absorption tests with patients' serums

Locality	Number of serums tested	Abortus infections		Melitensis infections	
		Number	Percent	Number	Percent
Charlotte, N. C.....	7	2	28.6	5	71.4
San Antonio, Tex.....	10	4	40	6	60
Kansas City, Kans.....	10	8	80	2	20

Five out of 7, or 71.4 percent, of serums from cases in the Charlotte (N. C.) area; six out of 10, or 60 percent, of serums from cases in the San Antonio (Tex.) area; and 2 out of 10, or 20 percent, of serums from the cases in the Kansas City (Kans.) area gave results indicating infection with the *melitensis* variety. At any rate, the infecting strains in these cases were not of the *abortus-suis* type. They were either *melitensis* infections or infections with atypical strains. Judging from the review of literature, the great majority of these cases must have been infected with the *melitensis* variety. In all of these cases the infection had been contracted in the State where the study was made.

DISCUSSION

It was to be expected that a large percentage of the cases of brucellosis in Texas would be found to be infected with the *melitensis* variety. It was surprising, however, to find that the majority of human cases studied in the North Carolina area and a considerable percentage of the cases in the Kansas area were infected with the *melitensis* variety.

Since comparatively few goats are raised in the United States outside of the Southwestern States, the spread of human brucellosis infections with the *melitensis* variety must depend largely on the susceptibility of cattle to this infection. Hence, the reports of cattle infected with the *melitensis* variety are of great interest.

On account of the much higher virulence of the *melitensis* variety for man, we can expect that whenever it infects cattle in any community in this country our experience will be the same as that in the east of France—the proportion of human infections with the *melitensis* variety to infections with the *abortus* variety will be far greater than the proportion between the two varieties incident in cattle. As in Argentina, we in this country may also expect a greater proportion of severe cases in regions where the *melitensis* variety exists.

Cultural studies are being made on some of the chronic cases in two of the survey areas, and it is hoped that the observations reported here may be extended by the study of strains.

SUMMARY

Human infections with *Brucella melitensis* variety *melitensis* have long been known in southwestern United States. In the literature are found records of occasional human and bovine infections with the *melitensis* variety in various other sections of the United States.

A review is given of the reports in which the grouping of *Brucella* according to serological reactions is correlated with the grouping according to bacteriostatic reactions. Excepting in certain restricted localities, there is a low percentage of atypical strains in which the groupings according to the two systems do not agree. Of 259 American strains which have been studied by various investigators, only 19 (7.3 percent) were atypical. Hence, although agglutinin absorption tests will not classify an individual *Brucella* strain in the *abortus-suis* or *melitensis* group with absolute certainty, collected data will give information as to the types of infections in a given locality.

The results of this study indicate that the percentages of human infections with the *melitensis* variety in the 3 survey areas were as follows: In Charlotte, N. C., 5 out of 7 brucellosis cases, or 71.4 percent; in San Antonio, Tex., 6 out of 10 cases, or 60 percent; in Kansas City, Kans., 2 out of 10 cases, or 20 percent.

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PULMONARY TUMORS IN MICE

II. The Influence of Heredity upon Lung Tumors Induced by the Subcutaneous Injection of a Lard-Dibenzanthracene Solution¹

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FREQUENCY OF OCCURRENCE

While primary lung tumors are not common in most species of animals, they are known to occur with exceptional frequency in mice. In fact, the prevalence of pulmonary growths in mice is one of the most striking features in the study of malignant tumors in this species. Livingood (12), in 1896, first described a primary lung tumor which arose in a bronchus of an albino mouse and which he diagnosed as an adenocarcinoma. Haaland (7) reported five instances of spontaneous lung tumors in mice, but Tyzzer (21, 22) was the first investigator to give these growths thorough consideration. He observed primary lung nodules in 12 mice ranging in age from 5½ months to "very old", among which were 5 white, 3 gray, 2 black, 1 brown, and 1 black and white animals. A detailed and extensive description of each of these tumors was given, and the conclusion was reached that most of them "correspond to a single type, although there are minor variations." Mitosis was observed in only one case, but in two cases the growths extended into bronchi and were undoubtedly malignant. It was found difficult "to decide what name to apply to this type of tumor", but they were finally designated as "papillary cyst-adenoma." A total of 83 spontaneous tumors in 70 mice was found, of which 62 percent were primary lung growths, and the conclusion was reached that, in mice, primary tumors appear to be more frequent in the lungs than in any other organ.

Tyzzer described two types of growth, the first being the "papillary cyst-adenoma", in which the epithelium is arranged in a single layer upon irregular folds of supporting tissue and resembling in certain respects the structure of the lung. The tumor cells are either columnar or cuboidal and have no cilia. He could not decide whether the tumors arose from the epithelium of the bronchi or alveoli, and stated that "in most cases it resembles the bronchial epithelium, but it sometimes resembles the thickened alveolar epithelium." The paucity of mitotic figures was noted, which indicated that the growth rate of such tumors must be very slow. He concluded that some of the tumors "must be considered carcinomata because of the irregular growth of the epithelium." The second type of growth was designated as an "epidermoid carcinoma" of the lung with diffuse growth of epithelium showing a tendency to keratinization. This type of growth is much

¹ From the Office of Cancer Investigations, U. S. Public Health Service, Harvard Medical School, Boston, Mass.

rarer than the papillary cyst-adenoma type. Tyzzer also called attention to the fact that the lungs of mice frequently exhibit areas of chronic inflammatory hyperplasia.

In addition to his descriptions of these growths, to which little has been added by subsequent observers, Tyzzer also discovered the influence of heredity upon their development. Starting with a female mouse with a lung tumor and a male free from tumor, he obtained 62 progeny which reached maturity, and of these 27 percent developed primary lung nodules.

Jobling (10, 11) found primary lung growths to be next in frequency to mammary gland carcinomas in a series of 26 mice exhibiting 41 spontaneous tumors of which 29 arose in the mammary glands and 9 in the lungs. Haaland (8) described 353 primary tumors in mice and, while not giving complete statistics, states that "the adenomatous tumours of the lung vie with the mammary tumours in frequency." His studies led him to conclude that a large proportion of the lung growths are "undoubtedly malignant", but in some cases "their exact nature is uncertain", since many appeared to be hypertrophic changes instead of malignant growths. Nodules occurred only in the lungs of old mice and were often associated with chronic inflammatory processes, which were noted frequently in the lungs of normal mice.

Slye, Holmes, and Wells (17) were the next investigators to comment upon the spontaneous lung tumors of mice. After reviewing the earlier literature, they concluded that these growths are peculiar not only in frequency but also in structure, when contrasted with pulmonary tumors of other species. They observed 160 mice bearing lung nodules in the first 6,000 autopsied mice of Slye's stock; lung tumors constituted one-third of all tumors found in the 6,000 autopsies and were next in frequency to mammary gland tumors. Only those nodules "that seem fairly entitled to be classed as tumors" were included, and in accord with Tyzzer and Haaland they commented upon the "many nodules caused by inflammatory hyperplasia." The authors presented an interesting table of lung tumors in their mice in which the tumors were tabulated as to their growth characteristics. Of the 160 nodules, 20 were classed as "unquestionable carcinomas", 43 as showing "a reasonably sure malignant tendency", 41 as of "doubtful malignancy", and 56 as "benign". The types of growth ranged from those which exhibited active infiltration and regional metastases in the lungs to the "benign" nodules which they regarded as true tumors and not "inflammatory hyperplasia."

The nodules usually appeared in mice over 1 year of age, and sex apparently had little influence on their occurrence. In addition, Slye, Holmes, and Wells were the first to observe the presence of metastases outside the lungs. Four such cases were reported, two exhibiting secondary growths in the mediastinal lymph nodes and

two in the mediastinal lymph nodes, chest wall, diaphragm, and kidney. So far as heredity is concerned, the statement was made that "Hereditary influences show a marked relation to the occurrence and character of these lung tumors." Hill (9) raised mice on varied beddings and diets. These factors had little influence on the origin of spontaneous tumors, but in 793 experimental and control mice he found 140 with spontaneous pulmonary growths.

Other investigators have noted the occurrence of primary lung tumors in mice, but it is believed that the reports referred to above are sufficient to establish the fact that in this species pulmonary growths appear with unusual frequency. It is necessary to remember that many of these observations were made in mice which had not been inbred and therefore may be regarded as presenting the incidence of lung tumors in laboratory mice. With the advent of studies pertaining to the effect of inbreeding on the occurrence of cancer in mice, it was only natural that investigators should concentrate on tumors which arose in sites easily accessible to macroscopic examination and the mammary glands were found to meet this requirement. However, the influence of heredity on pulmonary growths in mice has received attention from some investigators.

INFLUENCE OF HEREDITY

Lynch (13, 14, 15) has given this problem extensive consideration by recording the lung tumor incidence in two strains of mice. One of these, strain 1194, was inbred from the sixth to the fifteenth generation by brother to sister matings, "or rarely cousin by cousin", and consisted mostly of black agouti mice, although a few were pinkeyed or brown agouti. Lynch found a lung tumor rate of 6.7 percent in strain 1194 animals in a total of 208 mice that lived longer than 1 year. The youngest age at which a lung tumor was found at autopsy was 18 months. The other strain studied was Lynch's line of the Bagg albinos. In 135 individuals of this strain that lived to be at least 1 year old, Lynch recorded a lung tumor incidence of 37.04 percent. The youngest age at which a lung tumor was found at autopsy was 15 months. Crosses between these two strains produced 14 individuals of the first generation that lived 16 months or longer, and of these, 5, or 31 percent, developed lung tumors. Of the second outcross generation 49 mice lived to be more than 1 year of age, and of these, 11, or 22 percent, developed lung tumors. From these results Lynch suggested that lung tumor susceptibility is inherited as a dominant character.

Lynch also ascertained the incidence of lung nodules in the offspring of mice that had or did not have lung tumors. The results may be briefly summarized as follows: Lung tumors arose in 19 percent of the progeny of mice which were free from lung tumors when autopsied;

in 40 percent of the progeny of parents, one of which had lung tumor; and in 48 percent of the progeny of parents, both of which had lung tumors. Since pulmonary growths appeared in the mice whose parents were both free of lung tumor, Lynch suggested that "tumor susceptibility is not only dominant but variable." Other studies led her to conclude that sex had but little influence on the occurrence of lung tumors and that the lung growths were found most frequently in mice coming to autopsy when 2 years of age or older.

Lynch has presented further evidence that lung tumor susceptibility is inherited. A new stock of albino mice, designated as strain D, is introduced which had a lung tumor rate of 34 percent in mice over 6 months of age. A male mouse of strain D was bred to 6 females of strain 1194 and the 45 progeny which lived more than 6 months showed a lung tumor incidence of 24.4 percent. Male mice of the first generation were backcrossed to females of the parent stocks. When backcrossed to females of strain D, the progeny, numbering 404 individuals, gave a lung tumor incidence of 32.2 percent; but when the males were backcrossed to females of strain 1194, the progeny, numbering 218 mice, gave a lung tumor rate of 7.3 percent. Thus it was again indicated that the tendency to develop pulmonary growths is inherited. In other reports Lynch (16) has presented evidence to show that tar-painting may be utilized to demonstrate the inheritance of this organ susceptibility.

A strain of highly inbred mice with a high incidence of spontaneous pulmonary growths has been described by investigators of the Roscoe B. Jackson Memorial Laboratory. These mice designated as strain A have, according to Strong (19, 20), descended from the Bagg albino strain. Bittner (4, 5) states that 55 percent of males living 10 months or longer develop pulmonary growths, and of breeding females which develop mammary gland tumors 36 percent also have lung nodules. In the course of a crossbreeding experiment between this stock and another which does not give rise to lung tumors he found primary lung growths in the hybrid animals. Recently Bittner (6) has published a thorough study of the lung tumor incidence in this interesting strain of mice. Of 123 breeding females coming to autopsy, 26.1 percent had primary lung tumors. Of 126 virgin females, 77 percent developed primary lung growths, the average age at autopsy being 16.6 months. Of 116 breeding males, 71.6 percent exhibited primary pulmonary tumors, the average age at autopsy being 14.8 months. Thus, of 242 virgin females and breeding males of this strain, 181, or 74.7 percent, developed lung tumors spontaneously.

EXPERIMENTAL

Mice of strain A have been used in this laboratory (1, 2) in investigations of the appearance of lung tumors following subcutaneous

injections of a lard solution of 1, 2, 5, 6-dibenzanthracene. It has been found (3) that this carcinogenic compound acts similarly to tar in eliciting lung tumors in mice and that mice of strain A are excellent test animals for such experiments. The idea suggested itself that a cross-breeding experiment between strain A mice and a strain known to exhibit a low incidence, if any, of spontaneous lung growths might be of interest. Mice of the C 57 black strain were chosen as suitable (18) for the experiment. It was decided to inject a lard-dibenzanthracene solution subcutaneously into most of the experimental animals in order to test for susceptibility to lung tumors. This procedure should also yield some information as regards the influence, the age, sex, or color of the progeny might have on their susceptibility to the carcinogenic action of the compound in both the lungs and subcutaneous tissues.

RESULTS OF CROSS-BREEDING

Young adult mice of the pure strains A and C 57 black were mated on July 15, 1935, as follows: 21 females of strain A to males of strain C 57 black and 21 females of strain C 57 black to strain A males. There were 179 black offspring (96 females and 83 males) born between August 15 and October 5, 1935.

On October 30, 1935, the females of the first hybrid generation were mated to their brothers and, as the result of this mating, 665 mice were obtained. The sex and color of the second hybrid generation are summarized below:

	Females	Males	Total
Black.....	175	205	380
Albino.....	86	86	172
Brown.....	53	60	113
Total.....	314	351	665

DIBENZANTHRACENE INJECTIONS

A sufficient quantity of a lard-dibenzanthracene solution was made up to last throughout the course of the experiment in order to obviate any difference in results which might be attributed to different solutions. Each cubic centimeter of lard contained 4 milligrams of 1, 2, 5, 6-dibenzanthracene; the procedure for preparing the solution has been described (1) elsewhere.

On November 1, 1935, all living females (38) of both strain A and C 57 black, along with 55 males and 4 females of the first hybrid generation, each received 0.2 cc of the lard-dibenzanthracene solution in the subcutaneous tissue of the right axillary region. The injection was repeated on November 15, 1935.

On January 22, 1936, 92 females of the first hybrid generation which had been used as breeders and 370 mice of the second hybrid generation were available. Of these, 62 mice of the first hybrid and 305 of the second hybrid generations were each given 0.2 cc of the lard-dibenzanthracene solution in the right axilla. Thirty of the first and 65 of the second hybrid generations and litter mates of the injected mice were set aside as normal controls. The experimental mice received another 0.2 cc injection on February 5, 1936. The color and sex of the injected mice of the second hybrid generation were divided as follows:

	Female	Male	Total
Black.....	58	73	131
Albino.....	50	51	101
Brown.....	34	39	73
Total.....	142	163	305

RESULTS IN PURE STRAIN ALBINO AND C 57 BLACKS

Thirty-eight of these females received the first injection on November 1, 1935. The first tumor was found on February 12, 1936. Between the time of injection and the time of appearance of the first subcutaneous tumor, 15 of the strain A and 8 of the strain C 57 blacks had succumbed to an epidemic of *B. piliformis* (23). Lung tumors were found in one of the strain A mice; none of the others had tumors when autopsied. Of the 10 remaining C 57 black mice, 9 developed subcutaneous tumors and 1 died without any evidence of tumor. None had macroscopic lung nodules. Of the five strain A mice, four developed both subcutaneous and lung tumors. One died on March 3, 1936, without a subcutaneous tumor but with multiple lung nodules.

RESULTS IN THE FIRST HYBRID GENERATION

These were black mice, 55 males and 4 females, which received an initial lard-dibenzanthracene injection on November 1, 1935. Ten of the males died tumor-free before February 2, 1936, when the first subcutaneous tumor was noted. Of the remaining 49 mice, 47 developed subcutaneous tumors and 42 developed lung tumors; 1 mouse died without a tumor.

There were 62 black mothers of the second hybrid generation which received their first lard-dibenzanthracene injection on January 22, 1936. Only one of these had died when the first subcutaneous tumor appeared on April 23, 1936. Of the remaining 61 animals, 54 developed subcutaneous tumors and 57 developed lung tumors.

The last three mice were killed on November 25, 1936. None of these had a subcutaneous tumor, but the lungs of all three contained multiple lung growths. Up to July 23, 1936, 39 of this group developed subcutaneous tumors and had been autopsied; 35 of them exhibited multiple lung nodules. On July 23, 1936, 11 of the uninjected controls were killed and examined for the presence of macroscopic lung nodules; 10 were negative and 1 had a single lung nodule.

RESULTS IN THE SECOND HYBRID GENERATION

These animals received their first injection on January 22, 1936, and the first tumor arose on April 23, 1936, just three months later. Prior to April 23, 12 had died without any macroscopic tumor. The animals were examined once each week for the appearance of subcutaneous growths, and all were autopsied for the presence of lung tumors. Up to July 23, 1936, only 10 of these mice died from other causes; and of these, 5 had lung tumors only and 5 were negative in both subcutaneous tissue and lungs.

On July 23, 1936, there were 62 of these mice (17 males and 45 females) alive. All of them and 26 normal controls of the same hybrid generation were killed and autopsied. The findings in these 88 mice are summarized below:

	Injected mice	Control mice
No macroscopic tumor.....	8	25
Subcutaneous tumor only.....	1	0
Lung tumors only.....	43	1
Subcutaneous tumor and lung tumors.....	10	0

It is seen that 43 of the injected mice had lung tumors only, and it should be mentioned that of the 62 injected mice, 53 exhibited multiple lung tumors, while of the 26 controls only 1 had a single pulmonary growth.

A summary of the findings as regards the appearance of subcutaneous and lung tumors in all the first and second hybrid generations is presented in table 1.

TABLE 1.—*Subcutaneous and lung tumors in first and second hybrid generations following subcutaneous injections of a lard-dibenzanthracene solution*

	First hybrid generation	Second hybrid generation	First generation controls killed on 7/23/36	Second generation controls killed on 7/23/36
Died or killed without tumor.....	12	26	10	25
Subcutaneous tumors only.....	10	60	—	—
Lung tumors only.....	8	* 48	1	1
Both subcutaneous tumors and lung tumors.....	91	171	—	—

An analysis of the results in the second hybrid generation has thus far failed to reveal any influence exerted by color, sex, or pedigree upon the occurrence of the induced lung growths.

THE INFLUENCE OF SEX ON THE APPEARANCE OF SUBCUTANEOUS TUMORS

As stated previously, the mice were examined each week for the presence of subcutaneous tumors; and as the tumors arose, the mice were placed in other cages for observation or were killed and autopsied. As the experiment progressed, this procedure drew attention to the fact that cages containing male mice of the second hybrid generation were emptied earlier than those in which the females were kept. At the conclusion of the experiment the time of appearance of the subcutaneous tumors in both the first and second hybrid generations was tabulated, according to sex. The findings are summarized in table 2. Attention is directed to the first column of figures in the table, which indicates the number of mice alive at the time the first subcutaneous tumor arose and not the total number of mice injected. In the table the numbers of new tumors discovered each week are listed according to the sex of the animal in which they arose. Just below these figures and in heavy face type is the weekly percentage of the total number of mice of each sex developing subcutaneous tumors. It is seen that the males of both generations responded to the carcinogenic agent by developing tumors earlier than did the females.

TABLE 2.—Time in weeks of the appearance of dibenzanthracene tumors

Time in weeks.....		14	15	16	17	18	19	20	21	22	23	24	25	26	27	31	32	34	36	37	40	Total number of tumors	Died or killed without subsequent tumor	
Hybrid generation	Sex	(a) Numbers of new tumors and (b) total percentages of mice with tumor																						
First.....	Male.....	1	2	6	3	3	4	0	5	2	5	1	2	1	3	1	1	1	1	1	1	43	2	
	Female.....	2	15	21	28	38	58	67	71	82	84	89	93	95	96	96	96	96	96	96	96	96	96	7
Do.....	Male.....	3	6	14	17	20	23	34	37	43	52	57	58	63	71	77	80	83	86	88	89	129	18	
	Female.....	5	8	14	17	16	16	16	10	11	3	5	6	6	6	6	6	6	6	6	6	129	18	
Second.....	Male.....	3	4	10	17	28	39	50	61	68	75	77	80	84	87	90	91	92	93	94	95	102	44	
	Female.....	1	1	4	6	10	19	7	5	6	8	8	10	11	6	6	6	6	6	6	6	102	44	
Do.....		0.6	1	4	8	15	28	32	35	39	45	51	58	66	70	75	78	81	84	87	90	95	102	44

(a)

(b)

(c)

(d)

(e)

(f)

(g)

(h)

SUMMARY AND CONCLUSIONS

In this experiment 21 females of strain A were bred to males of strain C 57 black, and 21 females of strain C 57 black were bred to males of strain A. The mice of strain A are known to be susceptible to both spontaneous and induced lung tumors, while the mice of strain C 57 black are known to be very resistant to the development of all spontaneous growths, and in this laboratory no lung tumors have thus far been induced in them by the subcutaneous injection of a lard-dibenzanthracene solution. The progeny of this mating has been designated as the first hybrid generation. Females of the first hybrid generation were mated to their brothers to procure mice designated as of the second hybrid generation.

Most of the first hybrid generation were injected subcutaneously with a lard-dibenzanthracene solution. Out of 121 of the injected animals, 11 died without tumor before the appearance of the first subcutaneous tumor at the injection site. Practically all the remaining 110 mice were kept under observation for the occurrence of subcutaneous or lung tumors; the last 3 were killed 11 months after the initial subcutaneous injection. Of the 110 animals, 101, or 91.9 percent, developed a subcutaneous tumor and 99, or 90 percent, developed lung nodules.

Of the second hybrid generation, 305 mice of black, white, or brown coat color were injected subcutaneously with a lard-dibenzanthracene solution. Twelve of these died tumor-free before the appearance of the first subcutaneous tumor. The remaining 293 mice were kept under observation for 6 months, during which time 231 developed a subcutaneous tumor or died from other causes. At the end of the 6-month period the remaining 62 mice were killed and autopsied for subcutaneous and lung tumors. It was found that up to 6 months after the initial injection of these 293 mice, 231, or 78.8 percent, had developed a subcutaneous tumor, and 219, or 74.7 percent, had developed lung nodules.

The occurrence of all subcutaneous tumors in the two generations cannot be compared, because the first generation mice were kept for 11 months and the second generation mice kept for 6 months after the initial lard-dibenzanthracene injection. However, table 2 reveals that 88, or 80 percent, of the first generation had developed subcutaneous tumors 6 months after the first injection.

The first generation mice were hybrids from reciprocal crosses between a strain of mice in which the females exhibit a high incidence of spontaneous mammary tumors (strain A) and a line in which the females show a low incidence of mammary tumors (strain C 57 black). Investigators (18) at the Roscoe B. Jackson Memorial Laboratory have found that such reciprocal crosses reveal an extra-chromosomal

influence in the occurrence of spontaneous mammary gland tumors, for hybrids derived from females which belong to high tumor lines develop more mammary tumors than those derived from females belonging to low tumor lines. The findings in the first hybrid generation mice of this experiment indicate that the progeny from either outcross were equally susceptible to the carcinogenic action of dibenzanthracene. This was to be expected, for previous investigations (1) have shown that both high and low spontaneous mammary tumor lines are susceptible to the development of induced dibenzanthracene tumors.

So far as the occurrence of lung tumors is concerned, 90 percent of the first hybrid generation mice and 74.7 percent of the second hybrid generation developed these growths. The fact that 62 of the second generation mice were killed 6 months after injection, and before a subcutaneous tumor was present in most of them, had little influence on the percentage developing lung nodules, for it will be recalled that 53 of the 62 mice exhibited lung nodules when examined at autopsy.

The absence of lung growths in a high proportion of uninjected controls of both the first and second hybrid generations shows that the lung nodules in the injected mice did not arise spontaneously and, in addition, shows that the lungs of the 62 second generation mice killed on July 23, 1936, were more responsive to the induction of tumors than the subcutaneous tissues, for 43 had multiple lung tumors without any evidence of a subcutaneous growth. This finding is similar to the results obtained in other experiments (3) with strain A mice.

The presence of lung tumors in a high percentage of animals of both the first and second hybrid generations shows that the susceptibility of this organ to tumor formation induced by the subcutaneous injections of a lard-dibenzanthracene solution is inherited in a dominant manner.

In both hybrid generations the carcinogenic compound produced subcutaneous tumors earlier in the male mice. It is essential to note that the females of the first hybrid generation were injected approximately 3 months after their brothers had received their initial injection. Furthermore, practically all the females had raised a litter before their injections began. Hence, the age at the time of injection or the influence of breeding may have been of some significance in the later appearance of tumors in the females of this generation. These factors, however, cannot be held responsible for the difference which occurred in the second hybrid generation, for all of these females were virgins and all were injected at the same time as their male litter mates. Previous experiments in this laboratory have not revealed any such difference in susceptibility between the sexes. The reason for the results obtained with these outcross animals remains obscure.

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DEATHS DURING WEEK ENDED FEBRUARY 20, 1937

(From the Weekly Health Index, issued by the Bureau of the Census, Department of Commerce)

	Week ended Feb. 20, 1937	Correspond- ing week, 1936
Data from 85 large cities of the United States:		
Total deaths.....	10,403	10,445
Average for 3 prior years.....	9,475	
Total deaths, first 7 weeks of year.....	74,766	67,022
Deaths under 1 year of age.....	656	617
Average for 3 prior years.....	586	
Deaths under 1 year of age, first 7 weeks of year.....	4,504	3,965
Data from industrial insurance companies:		
Policies in force.....	69,207,100	67,058,356
Number of death claims.....	16,541	14,938
Death claims per 1,000 policies in force, annual rate.....	12.5	11.5
Death claims per 1,000 policies, first 7 weeks of year, annual rate.....	11.6	10.8

PREVALENCE OF DISEASE

No health department, State or local, can effectively prevent or control disease without knowledge of when, where, and under what conditions cases are occurring

UNITED STATES

CURRENT WEEKLY STATE REPORTS

These reports are preliminary, and the figures are subject to change when later returns are received by the State health officers

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Feb. 27, 1937, and Feb. 29, 1936

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936
New England States:								
Maine.....			212	5	6	255	0	0
New Hampshire.....	1			1	76	13	0	0
Vermont.....						496	0	0
Massachusetts.....	4	8			768	916	4	12
Rhode Island.....	1				201	43	2	1
Connecticut.....	2	4	169	17	474	91	2	2
Middle Atlantic States:								
New York.....	31	51	145	178	439	2,636	11	27
New Jersey.....	9	16	134	62	1,190	159	6	7
Pennsylvania.....	45	41			219	797	7	6
East North Central States:								
Ohio.....	38	35	447	127	99	421	13	12
Indiana.....	9	27	133	48	11	40	3	1
Illinois.....	37	39	162	42	36	28	7	16
Michigan.....	16	7	4	10	52	44	2	4
Wisconsin.....	1	2	220	64	14	84	1	3
West North Central States:								
Minnesota.....	6	3	1	2	32	289	2	1
Iowa.....	2	15	8	6	2	4	0	3
Missouri.....	19	19	944	650	8	20	3	12
North Dakota.....	1	5	7	12	2		0	0
South Dakota.....	1	3	9	2			1	2
Nebraska.....	5	9	30		13	29	0	0
Kansas.....	9	15		32	6	12	0	3
South Atlantic States:								
Delaware.....	1		7		76	66	0	1
Maryland.....	8	9	372	72	554	146	2	11
District of Columbia.....	10	22	28	2	75	25	1	7
Virginia.....	14	11			269	86	14	48
West Virginia.....	14	12	1,252	218	1	21	10	9
North Carolina.....	25	16	173	482	64	55	3	8
South Carolina.....	3	1,346	1,509	54	12		2	16
Georgia.....	8	6	1,262	1,819			3	9
Florida.....	9	8	35	33	5	6	0	2
East South Central States:								
Kentucky.....	13	16	493	80	243	73	17	42
Tennessee.....	11	14	844	338	10	52	10	0
Alabama.....	37	28	1,546	2,383	26	5	8	1
Mississippi.....	7	4					2	3

See footnotes at end of table.

*Cases of certain communicable diseases reported by telegraph by State health officers
for weeks ended Feb. 27, 1937, and Feb. 29, 1936—Continued*

Division and State	Diphtheria		Influenza		Measles		Meningococcus meningitis	
	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936
West South Central States:								
Arkansas.....	11	3	980	140	-----	2	3	3
Louisiana.....	12	15	389	78	8	51	2	3
Oklahoma.....	7	10	974	256	12	13	7	10
Texas.....	29	15	3,480	655	310	418	14	5
Mountain States:								
Montana.....	2	1	132	45	2	16	0	1
Idaho.....	2	-----	67	3	34	15	1	0
Wyoming.....	-----	1	50	-----	2	8	0	0
Colorado.....	6	4	-----	-----	6	9	1	2
New Mexico.....	4	9	167	8	62	18	0	1
Arizona.....	5	5	269	304	239	37	2	0
Utah.....	-----	1	-----	-----	26	1	0	1
Pacific States:								
Washington.....	4	1	5	4	43	261	0	1
Oregon.....	1	2	196	267	7	733	1	0
California.....	21	33	1,915	1,661	110	1,890	13	11
Total.....	498	548	18,507	11,515	5,886	10,396	180	307
First 8 weeks of year.....	4,584	5,218	200,415	48,179	37,714	54,856	1,247	1,645

Division and State	Poliomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936
New England States:								
Maine.....	0	1	15	14	0	0	0	0
New Hampshire.....	0	0	23	3	0	0	0	0
Vermont.....	0	0	10	19	0	0	0	1
Massachusetts.....	0	0	255	285	0	0	1	3
Rhode Island.....	0	0	56	35	0	0	0	0
Connecticut.....	0	1	88	89	0	0	2	1
Middle Atlantic States:								
New York.....	0	1	953	1,277	4	0	2	6
New Jersey.....	0	1	174	520	0	0	1	3
Pennsylvania.....	0	1	561	512	0	0	3	1
East North Central States:								
Ohio.....	1	0	493	491	7	0	3	5
Indiana.....	1	0	216	344	8	1	0	8
Illinois.....	1	0	582	969	40	6	4	2
Michigan.....	0	0	771	297	3	0	1	1
Wisconsin.....	0	0	349	615	4	15	1	0
West North Central States:								
Minnesota.....	0	0	169	372	2	3	1	1
Iowa.....	2	0	351	106	35	20	0	3
Missouri.....	0	0	292	219	46	17	0	2
North Dakota.....	0	0	47	124	22	17	1	0
South Dakota.....	0	0	73	62	5	31	1	1
Nebraska.....	0	0	106	238	0	23	0	0
Kansas.....	0	0	378	325	22	47	1	1
South Atlantic States:								
Delaware.....	0	0	4	9	0	0	1	0
Maryland.....	0	0	51	98	0	0	1	3
District of Columbia.....	0	0	21	30	0	0	1	1
Virginia.....	0	1	35	57	0	0	1	2
West Virginia.....	0	0	55	44	0	1	3	1
North Carolina.....	1	1	33	34	0	0	8	0
South Carolina.....	0	0	6	5	0	0	3	0
Georgia.....	0	0	14	24	0	1	2	1
Florida.....	2	0	6	4	0	0	1	6

See footnotes at end of table.

Cases of certain communicable diseases reported by telegraph by State health officers for weeks ended Feb. 27, 1937, and Feb. 29, 1936—Continued

Division and State	Pollomyelitis		Scarlet fever		Smallpox		Typhoid fever	
	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936	Week ended Feb. 27, 1937	Week ended Feb. 29, 1936
East South Central States:								
Kentucky.....	0	1	65	76	0	0	2	0
Tennessee.....	0	0	18	24	0	0	4	1
Alabama ¹	2	3	12	30	0	0	1	5
Mississippi ²	0	0	12	14	0	0	2	1
West South Central States:								
Arkansas.....	0	1	11	9	5	1	0	4
Louisiana.....	1	0	14	19	0	3	6	3
Oklahoma ³	2	1	41	22	6	8	0	2
Texas.....	2	0	77	38	2	0	20	4
Mountain States:								
Montana.....	0	0	47	137	29	8	0	1
Idaho.....	0	0	25	92	1	4	0	1
Wyoming.....	0	0	36	127	3	1	0	0
Colorado.....	0	0	45	154	1	8	0	2
New Mexico.....	0	0	35	112	0	0	0	4
Arizona.....	0	0	16	34	0	2	1	2
Utah ⁴	0	0	23	143	0	1	0	0
Pacific States:								
Washington.....	0	0	63	81	5	13	2	1
Oregon.....	1	1	23	34	24	1	0	0
California.....	0	3	219	410	9	1	1	4
Total.....	16	17	6,969	8,777	283	233	82	88
First 8 weeks of year⁵.....	181	141	50,571	60,128	2,364	1,688	885	786

¹ New York City only.

² Week ended earlier than Saturday.

³ Typhus fever, week ended Feb. 27, 1937, 16 cases, as follows: South Carolina, 3; Georgia, 9; Alabama, 4.

⁴ Exclusive of Oklahoma City and Tulsa.

⁵ Figures for 8 weeks ended Feb. 27, 1937, include delayed reports.

SUMMARY OF MONTHLY REPORTS FROM STATES

The following summary of cases reported monthly by States is published weekly and covers only those States from which reports are received during the current week:

State	Menin- gococ- cus menin- gitis	Diph- theria	Infl- uenza	Mala- ria	Mea- sles	Pel- lagra	Polio- mye- litis	Scarlet fever	Small- pox	Ty- phoid fever
<i>January 1937</i>										
Arizona.....	10	16	3,333	3	602	-----	1	94	0	3
California.....	30	147	19,447	3	356	5	14	1,875	40	25
Kansas.....	8	43	14,943	-----	35	1	2	1,346	120	3
Mississippi.....	2	31	13,872	1,605	986	144	1	48	-----	21
Montana.....	9	12	10,600	-----	10	-----	0	221	61	5
North Dakota.....	6	3	966	-----	139	-----	0	260	145	1
Rhode Island.....	3	3	43	-----	663	-----	0	231	0	1
Vermont.....	-----	-----	215	-----	15	-----	0	39	0	3
Wisconsin.....	7	14	6,110	-----	84	-----	2	1,265	58	4

Summary of monthly reports from States—Continued

January 1937		January 1937—Continued		January 1937—Continued	
	Cases		Cases		Cases
Chicken pox:		Hookworm disease:		Septic sore throat—Contd.	
Arizona.....	263	Mississippi.....	244	Rhode Island.....	1
California.....	3,415	Impetigo contagiosa:		Wisconsin.....	18
Kansas.....	466	Kansas.....	1	Tetanus:	
Mississippi.....	791	Montana.....	4	California.....	3
Montana.....	236	Jaundice, epidemic:		Rhode Island.....	1
North Dakota.....	163	California.....	2	Trachoma:	
Rhode Island.....	238	Kansas.....	1	Arizona.....	21
Vermont.....	453	Leprosy:		California.....	8
Wisconsin.....	2,362	California.....	2	Mississippi.....	9
Dengue:		Mumps:		Montana.....	1
Mississippi.....	1	Arizona.....	118	Tularaemia:	
Dysentery:		California.....	2,663	California.....	1
Arizona.....	6	Kansas.....	739	Kansas.....	1
California (amoebic).....	6	Mississippi.....	825	Wisconsin.....	1
California (bacillary).....	13	Montana.....	296	Typhus fever:	
Kansas (bacillary).....	1	North Dakota.....	234	California.....	1
Mississippi (amoebic).....	59	Rhode Island.....	12	Undulant fever:	
Mississippi (bacillary).....	179	Vermont.....	134	Arizona.....	2
Montana (amoebic).....	1	Wisconsin.....	730	California.....	5
Encephalitis, epidemic or		Ophthalmia neonatorum:		Kansas.....	6
lethargic:		California.....	2	Mississippi.....	1
California.....	4	Mississippi.....	5	Vermont.....	4
Montana.....	2	Paratyphoid fever:		Wisconsin.....	1
North Dakota.....	1	California.....	1	Vincent's infection:	
Wisconsin.....	1	Puerperal septicemia:		Kansas.....	5
Food poisoning:		Mississippi.....	20	North Dakota.....	5
California.....	41	Rabies in animals:		Whooping cough:	
German measles:		California.....	106	Arizona.....	26
Arizona.....	21	Mississippi.....	30	California.....	1,278
California.....	89	Scabies:		Kansas.....	107
Kansas.....	6	Kansas.....	4	Mississippi.....	209
Montana.....	13	Montana.....	2	Montana.....	19
Rhode Island.....	1	Septic sore throat:		North Dakota.....	4
Vermont.....	12	Arizona.....	1	Rhode Island.....	132
Wisconsin.....	3	California.....	18	Vermont.....	115
Granuloma, coccidioidal:		Kansas.....	3	Wisconsin.....	439
California.....	4	Montana.....	17		

WEEKLY REPORTS FROM CITIES

City reports for week ended Feb. 20, 1937

This table summarizes the reports received weekly from a selected list of 140 cities for the purpose of showing a cross section of the current urban incidence of the communicable diseases listed in the table. Weekly reports are received from about 700 cities, from which the data are tabulated and filed for reference.

State and city	Diph- theria cases	Influenza		Meas- les cases	Pneu- monia deaths	Scar- let fever cases	Small- pox cases	Tuber- culo- sis deaths	Ty- phoid fever cases	Whoop- ing cough cases	Deaths, all causes
		Cases	Deaths								
Maine:											
Portland.....	0	3	0	0	9	1	0	1	0	3	27
New Hampshire:											
Concord.....	0		3	0	4	2	0	0	0	0	14
Manchester.....	0		4	0	7	0	0	0	0	0	29
Nashua.....	0			0	7	0	0		0	0	
Vermont:											
Barre.....	0		1	0	0	1	0	1	0	6	2
Burlington.....	0		0	0	0	6	0	0	0	0	7
Rutland.....	0		0	0	3	1	0	0	0	0	11
Massachusetts:											
Boston.....	3		5	10	60	52	0	9	1	146	312
Fall River.....	2		0	10	8	3	0	1	0	2	43
Springfield.....	0		0	27	2	1	0	2	0	10	43
Worcester.....	0		0	187	11	6	0	3	0	36	66
Rhode Island:											
Pawtucket.....											
Providence.....	0	14	4	203	13	44	0	4	0	10	86
Connecticut:											
Bridgeport.....	0	9	2	43	6	18	0	2	0	0	43
Hartford.....	0	32	0	2	12	10	0	1	0	6	64
New Haven.....	1	17	1	0	3	5	0	0	0	0	60
New York:											
Buffalo.....	0		2	42	29	25	11	7	0	40	201
New York.....	48	74	14	151	168	457	0	98	4	112	1,617
Rochester.....	0	9	3	0	8	3	0	1	0	11	94
Syracuse.....	0		1	32	15	70	0	1	0	22	71

City reports for week ended Feb. 20, 1937—Continued

State and city	Diphtheria cases	Influenza		Measles cases	Pneumonia deaths	Scarlet fever cases	Small-pox cases	Tuberculosis deaths	Typhoid fever cases	Whooping cough cases	Deaths, all causes
		Cases	Deaths								
New Jersey:											
Camden.....	1		2	2	4	3	0	0	0	13	35
Newark.....	0	7	2	450	14	12	0	9	0	29	110
Trenton.....	0	6	2	2	6	6	0	4	0	0	54
Pennsylvania:											
Philadelphia.....	7	35	20	12	60	291	0	25	0	101	630
Pittsburgh.....	2	14	8	15	29	50	0	10	0	36	178
Reading.....	0		0	3	5	3	0	3	0	22	29
Scranton.....	1			0		19	0		0	3	
Ohio:											
Cincinnati.....	3	5	2	33	18	21	0	10	0	7	183
Cleveland.....	2	149	11	4	34	76	0	11	0	57	240
Columbus.....	2	119	13	3	12	11	0	10	0	0	128
Toledo.....	0	11	8	5	8	11	0	3	0	35	92
Indiana:											
Anderson.....	0		1	2	1	12	0	1	0	1	19
Fort Wayne.....	0		0	0	6	1	0	5	0	2	28
Indianapolis.....	0		5	1	28	45	0	5	0	22	119
South Bend.....	0		1	0	3	6	0	0	0	3	25
Terre Haute.....	1		0	0	0	3	0	0	0	0	38
Illinois:											
Alton.....	0		0	0	1	4	0	1	0	0	10
Chicago.....	13	29	4	14	82	243	0	33	2	63	800
Elgin.....	0		0	0	1	1	0	0	0	13	10
Moline.....	0		0	0	3	0	0	2	0	12	12
Springfield.....	0		1	0	6	4	0	0	0	6	22
Michigan:											
Detroit.....	9	4	3	8	32	449	0	18	0	91	277
Flint.....	1		1		3	22	0	0	0	3	32
Grand Rapids.....	0		1	8	2	15	0	1	0	17	48
Wisconsin:											
Kenosha.....	0		0	0	0	4	0	1	0	3	4
Madison.....	0		0	1	0	11	0	0	0	2	13
Milwaukee.....	0	3	3	2	15	72	0	4	0	21	138
Racine.....	0		0	0	1	6	0	0	0	0	12
Superior.....	0		1	1	1	3	0	0	0	5	14
Minnesota:											
Duluth.....	0		2	0	0	23	0	0	0	3	30
Minneapolis.....	1		0	2	20	26	0	1	0	21	109
St. Paul.....	1		0	1	7	15	0	2	0	42	61
Iowa:											
Cedar Rapids.....	0			0		2	0		0	0	
Davenport.....	0			0		3	0		0	0	
Des Moines.....	1			0		41	0		0	0	39
Sioux City.....	0			0		27	0		0	2	
Waterloo.....	0			0		23	0		0	9	
Missouri:											
Kansas City.....	1	7	4	4	6	84	0	1	0	4	101
St. Joseph.....	1		1	0	7	10	24	2	0	1	56
St. Louis.....	9		4	1	18	50	2	5	0	69	228
North Dakota:											
Fargo.....	0		0	1	0	8	0	0	0	0	3
Grand Forks.....	0			0		0	0		0	0	
Minot.....	0		0	0	0	0	1	0	0	0	9
South Dakota:											
Aberdeen.....	0			0		5	0		0	0	
Nebraska:											
Omaha.....	0		0	0	14	6	0	4	0	4	87
Kansas:											
Lawrence.....	0		0	0	2	0	0	0	0	0	5
Topeka.....	0		1	0	11	12	0	0	0	0	27
Wichita.....	0	2	2	2	7	10	0	1	0	0	33
Delaware:											
Wilmington.....	0		2	32	8	4	0	0	0	2	40
Maryland:											
Baltimore.....	8	35	4	362	51	17	0	15	0	63	273
Cumberland.....	0	1	1	0	3	0	0	0	0	0	14
Frederick.....	0		0	0	2	0	0	0	0	0	4
Dist. of Col.:											
Washington.....	5	27	10	63	34	23	0	14	1	18	220
Virginia:											
Lynchburg.....	1		1	3	3	0	0	2	0	2	15
Norfolk.....	0	23	0	0	9	5	0	0	0	0	34
Richmond.....	0		6	4	11	1	0	1	0	1	61
Roanoke.....	1		0	72	3	1	0	2	0	0	19

City reports for week ended Feb. 20, 1937—Continued

State and city	Diph- theria cases	Influenza		Mea- sles cases	Pneu- monia deaths	Scar- let fever cases	Small- pox cases	Tuber- culosis deaths	Ty- phoid fever cases	Whoop- ing cough cases	Deaths, all causes
		Cases	Deaths								
West Virginia:											
Charleston.....	0	9	0	0	8	1	0	1	0	0	25
Huntingdon.....	1			0		2	0	0	0	0	
Wheeling.....	0		4	1	5	5	0	0	0	0	39
North Carolina:											
Gastonia.....	0	1	0	0	0	0	0	0	0	0	
Raleigh.....	0		0	1	0	0	0	1	0	1	21
Wilmington.....	1		0	0	1	0	0	1	0	0	7
Winston-Salem.....	0	1	0	1	5	3	0	2	0	1	20
South Carolina:											
Charleston.....	1	174	4	0	4	2	0	0	2	0	31
Columbia.....	0		0	0	4	0	0	1	0	0	26
Florence.....	0		0	0	3	0	0	0	0	0	17
Georgia:											
Atlanta.....	1	443	9	0	12	1	0	3	0	2	98
Brunswick.....	0	1	1	0	0	0	0	0	0	0	5
Savannah.....	1	120	2	0	4	2	0	5	0	3	40
Florida:											
Miami.....	1	12	1	2	1	2	0	2	0	1	39
Tampa.....	3	2	1	1	1	2	0	1	0	0	22
Kentucky:											
Ashland.....	0	30	0	0	3	0	0	0	0	0	11
Covington.....	0		1	0	1	0	0	1	0	0	18
Lexington.....	0	4	4	12	7	1	0	2	0	0	26
Tennessee:											
Knoxville.....	3	9	5	3	6	3	0	2	0	2	37
Memphis.....	0		20	1	29	0	0	7	0	34	146
Nashville.....	0		4	0	7	3	0	3	0	4	65
Alabama:											
Birmingham.....	2	201	9	0	7	5	0	4	0	5	75
Mobile.....	0	15	5	0	9	0	0	3	1	0	39
Montgomery.....	1	6		0		1	0		0	0	
Arkansas:											
Fort Smith.....	0			0		4	0		0	0	
Little Rock.....	0		1	0	14	1	0	8	0	0	25
Louisiana:											
Lake Charles.....	1		0	0	2	0	0	0	0	0	
New Orleans.....	9	75	10	0	35	2	0	12	0	0	192
Shreveport.....	0		2	0	10	0	0	2	1	0	43
Oklahoma:											
Muskogee.....	0			0		2	0		0	0	
Tulsa.....	0			0		5	0		0	9	
Texas:											
Dallas.....	4	24	24	8	19	13	0	3	0	11	115
Fort Worth.....	0		3	78	10	1	1	3	0	1	54
Galveston.....	0		0	0	7	1	0	0	0	0	33
Houston.....	2		3	0	26	1	0	4	1	0	98
San Antonio.....	0		15	7	17	0	0	9	0	1	100
Montana:											
Billings.....	0		0	0	1	1	0	0	0	0	7
Great Falls.....	0		3	0	5	0	1	0	0	0	11
Helena.....	0	72	0	17	3	18	0	0	0	0	5
Missoula.....	0		0	0	2	1	0	0	0	0	8
Idaho:											
Boise.....	0		0	0	1	0	0	1	0	0	10
Colorado:											
Colorado Springs.....	0		1	0	2	12	0	1	0	0	14
Denver.....	0		3	6	7	11	0	8	0	45	13
Pueblo.....	1		0	1	2	1	0	0	0	0	7
New Mexico:											
Albuquerque.....	0	38	0	0	0	6	0	3	0	9	9
Utah:											
Salt Lake City.....	2		1	8	6	10	0	0	0	8	41
Nevada:											
Reno.....											
Washington:											
Seattle.....	0		4	1	12	5	0	5	0	3	111
Spokane.....	0	4	4	0	8	3	0	0	0	4	45
Tacoma.....	1		2	0	4	6	0	0	0	0	43
Oregon:											
Portland.....	0	6	4	2	8	7	3	0	0	2	85
California:											
Los Angeles.....	13	155	18	24	75	35	0	29	0	65	490
Sacramento.....	1	134	3	1	7	5	0	3	0	1	41
San Francisco.....	3	87	10	1	14	31	0	9	0	20	210

City reports for week ended Feb. 20, 1937—Continued

State and city	Meningococcus meningitis		Polio-myelitis cases	State and city	Meningococcus meningitis		Polio-myelitis cases
	Cases	Deaths			Cases	Deaths	
Massachusetts:				Maryland:			
Boston.....	3	2	0	Baltimore.....	4	2	0
Rhode Island:				District of Columbia:			
Providence.....	1	0	0	Washington.....	2	1	0
New York:				Georgia:			
New York.....	9	5	0	Atlanta.....	1	0	0
Rochester.....	1	0	0	Kentucky:			
Syracuse.....	1	1	0	Ashland.....	1	0	0
Pennsylvania:				Louisville.....	4	5	0
Philadelphia.....	1	1	0	Alabama:			
Pittsburgh.....	2	0	0	Birmingham.....	1	0	0
Ohio:				Arkansas:			
Cincinnati.....	2	1	0	Little Rock.....	0	2	0
Cleveland.....	1	0	0	Texas:			
Illinois:				Dallas.....	1	0	0
Chicago.....	2	0	0	Fort Worth.....	2	1	0
Michigan:				Galveston.....	1	0	0
Detroit.....	1	0	0	Houston.....	3	0	0
Flint.....	1	1	0	Montana:			
Minnesota:				Billings.....	1	0	0
Minneapolis.....	1	1	0	Colorado:			
St. Paul.....	1	0	0	Denver.....	2	1	0
Missouri:				California:			
St. Louis.....	2	1	0	Los Angeles.....	2	5	1
Kansas:				Sacramento.....	1	0	0
Wichita.....	1	0	0	San Francisco.....	2	0	0

Encephalitis, epidemic or lethargic.—Cases: New York, 1; Philadelphia, 1; Flint, 1; Washington, D. C., 1; Louisville, 1.

Pellagra.—Cases: Baltimore, 1; Winston-Salem, 2; Charleston, S. C., 1; Savannah, 2; Birmingham, 1; Mobile, 1; Los Angeles, 1; San Francisco, 1.

Rabies in man.—Deaths: Atlanta, 1.

Typhus fever.—Cases: New York, 2; Savannah, 3; Miami, 2.

FOREIGN AND INSULAR

AUSTRIA

Vital statistics—1935.—The following table shows the number of marriages, births, and deaths in Austria for the year 1935:

Population.....	6,760,631	Deaths from—Continued:	
Marriages.....	45,375	Influenza.....	1,404
Births.....	91,111	Malaria.....	6
Total deaths.....	92,390	Measles.....	163
Deaths from:		Scarlet fever.....	103
Accidents.....	2,313	Sepsis.....	913
Cancer and other malignant tumors..	11,854	Suicide.....	2,506
Congenital debility.....	1,807	Syphilis.....	406
Diabetes.....	783	Tuberculosis.....	7,343
Diphtheria.....	992	Typhoid fever and paratyphoid fever..	121
Heart disease.....	15,365	Whooping cough.....	209

CUBA

Habana—Communicable diseases—4 weeks ended February 13, 1937.—During the 4 weeks ended February 13, 1937, certain communicable diseases were reported in Habana, Cuba, as follows:

Disease	Cases	Deaths	Disease	Cases	Deaths
Diphtheria.....	17	1	Poliomyelitis.....	12	
Leprosy.....	1		Scarlet fever.....	1	
Malaria.....	134	3	Tuberculosis.....	14	3
Measles.....		1	Typhoid fever.....	140	4

¹ Includes imported cases.

GERMANY

Vital statistics—Third quarter 1936.—Following are vital statistics for Germany for the third quarter of 1936:

Number of marriages.....	147,055	Number of deaths.....	172,394
Number of marriages per 1,000 population..	8.7	Number of deaths per 1,000 population....	10.2
Number of births.....	312,162	Deaths under 1 year of age.....	18,824
Number of births per 1,000 population.....	18.5	Deaths under 1 year of age per 100 live births..	5.9
Number of stillbirths.....	7,739		

ITALY

Vital statistics—1936.—Following are vital statistics for Italy for the year 1936:

Number of marriages.....	310,822	Number of live births per 1,000 population..	22.2
Number of marriages per 1,000 population..	7.2	Total deaths.....	582,612
Number of live births.....	955,189	Deaths per 1,000 population.....	13.5

CHOLERA, PLAGUE, SMALLPOX, TYPHUS FEVER, AND YELLOW FEVER

NOTE.—A table giving current information of the world prevalence of quarantinable diseases appeared in the PUBLIC HEALTH REPORTS for February 26, 1937, pages 255-267. A similar cumulative table will appear in the PUBLIC HEALTH REPORTS to be issued March 26, 1937, and thereafter, at least for the time being, in the issue published on the last Friday of each month.

Cholera

India—Bassein.—During the week ended February 20, 1937, 4 cases of cholera were reported in Bassein, India.

Plague

Hawaii Territory—Island of Hawaii—Hamakua District—Paauhau Sector.—A rat found March 1, 1937, in Paauhau Sector, Hamakua District, Island of Hawaii, Hawaii Territory, has been proved plague infected.

Smallpox

Siam—Tak Province.—During the week ended February 20, 1937, 26 cases of smallpox were reported in Tak Province, Siam.

Typhus Fever

Trans-Jordan—Kerak District.—During the week ended February 20, 1937, 3 cases of typhus fever were reported in Kerak District, Trans-Jordan.

Yellow Fever

Gold Coast—Accra.—On February 10, 1937, 2 cases of yellow fever were reported at Accra, Gold Coast.

Ivory Coast.—During the week ended February 20, 1937, yellow fever was reported in Ivory Coast as follows: 3 cases in Adzope, Agneby Circle, and 1 suspected case in Nzimcomoe Circle.